In December 1972 the CIBA Foundation held a symposium on the law and ethics of donor artificial insemination. The conclusion was that "AID is here to stay"[27] and that the law has got to consider AID not in a prohibitory way and perhaps only in a regulatory way so far as is required to make the technique acceptable to society.

We thank Miss D S Gosling for secretarial help.

ADDENDUM—Since this paper was written, further pregnancies have been achieved. Fifty pregnancies have resulted among the 107 patients who have completed the treatment schedule. This has increased the success rate by 2% to 46·7%

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Personal Paper

Landry Guillain-Barré syndrome: personal experience of acute ascending paralysis

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On a Saturday afternoon in May 1964, while watching the FA cup final on television, I developed headache, became feverish, and was, I thought, suffering from influenza. The next day I developed photophobia and the headache persisted. These symptoms were accompanied by intense cramp-like pains in the thighs, calves, and the soles of my feet. The pain was intense during the night and sleep became virtually impossible. At times I involuntarily cried out with pain.

As my condition did not improve during the next two weeks, I was admitted to St George's Hospital, Tooting, for investigation and treatment. Infectious mononucleosis was diagnosed. I was thought to have had a lymphocytic encephalitis, the pains being due to affected posterior nerve roots.

During the next 10 years I suffered pain, mostly in the shoulders, thighs, calves, and soles of the feet, usually in the afternoons and evenings. I also had headaches or abdominal pain, and there were days when I felt extremely lethargic. These attacks became less common and less painful over the years. But in October 1974 the attacks became more severe.

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An examination showed no clinical abnormality, and blood studies gave normal results.

Onset of Guillain-Barré syndrome

On Sunday 10 November 1974 I was driving to see a patient when pains in the shoulders forced me to take two paracetamol tablets. The consultation was complex and prolonged and I did not return home until 8 pm. That night sleep proved impossible because of pains mainly in the legs and feet. A bath at 3 am brought no relief, and by 5.30 am I was up and taking my dog for a walk. I experienced paraesthesiae in both feet and became apprehensive. I went to work as usual but have since been told that my gait was awkward and unsteady. When I went to see my doctor at about 5 pm I felt mildly unsteady with stiff calves. He found only very sluggish tendon reflexes in the legs and Rombergism but he arranged a consultation with a neurologist. I returned to work, but that night I again had little sleep.

Next morning the paraesthesiae had spread to both hands, in the distribution of the ulnar nerve. I drove to work but walking was awkward, my balance was uncertain, and I had to use a stick. By mid-morning I felt so unwell that the prospect of a busy outpatient clinic was too daunting and a colleague agreed to do it for me. I returned home during the afternoon feeling generally weak and unwell and was in bed by 6.30 pm. After that I remember only very severe pains, my wife ringing our general practitioner, and being given an injection of heroin and intravenous diazepam.

The next morning I recall telephoning the neurologist, who told me that I should be admitted to hospital immediately since he thought that I had the Guillain-Barré syndrome. Events seemed to

move fast. I remember the ambulance crew carrying me downstairs and the start of the journey. I had difficulty with starting micturition before getting into bed at the hospital, which made me realise that I had been having some difficulty for a week or so. I had to be helped into bed because I was weak and grossly ataxic in both arms and legs. I remember being seen by an anaesthetist and having my respiratory capacity measured, though I had felt no respiratory difficulty.

Physical examination confirmed widespread peripheral polyneuritis affecting motor and sensory systems in all limbs. My hands were severely affected though I retained some use of my arms, but my legs were completely paralysed. Sensory loss was almost complete, proprioceptive loss being the most obvious. Lumbar puncture confirmed that I had the Guillain-Barré syndrome.

Pain

The paralysis, weakness, and ataxia were frightening enough, but perhaps the most devastating thing was the pain, which was periodic and excruciating and always worst at night.

The next few weeks were unpleasant. I had an indwelling catheter and no knowledge of bowel action. The severe pain continued. Several times I experienced a disturbed body image: I was convinced that my left leg was sticking up at an angle of about 45°. I could see my paralysed leg lying on the bed, but the sensation was so clear and so painful that more than once I asked nurses to pull my leg down for me. One vivid and distorted sensation occurred when I felt I was lying across two commodes. I could even "see" the pattern on the lid of one and "feel" the edge of each. My arms were too ataxic to try to feel the edge with my fingers and any way I had no sensory perception so had to ask the nurse what I was lying on, which brought the reponse, "Nothing but the water bed."

These distortions of body image, bizarre sensory disturbances, and repeated vivid memories made nights especially long and unpleasant. Like a drowning man I saw events in my life pass before me. Repeatedly I experienced a sense of utter failure, and I starkly relived errors, omissions, and failures as husband, father, and in other spheres.

Treatment

I was aware of little specific treatment, although my doctors always discussed it with me. I was given steroids and developed a chest infection and a synovitis of the left hip, both of which proved painful, especially the left hip. I was too ataxic to feed myself but could sometimes drink from a baby's feeding mug. About three weeks after admission to hospital physiotherapy was begun. I felt a tremendous urge not to waste a second of the time available in the physiotherapy department, yet my muscles were so poor that I could not make them work.

Five weeks after admission I was clearly on the mend. I could feed myself crudely, use an electric shaver, clean my teeth, and ring a bell to summon help. With ataxic and wasted hands this is more difficult than one might think. Night pains remained severe and I needed two or three intramuscular injections of pentazocine every night for many weeks.

I was transferred to the local hospital, which is run by general practitioners but has full consultant cover and a most efficient physiotherapy department. From my admission there on 17 December 1974 until my discharge on 26 April 1975 not a day in hospital passed without at least one session with a physiotherapist—Christmas Day, Boxing Day, New Year's Day, and Good Friday included. A frame was fixed over my bed and the sister and her nursing staff arranged a second session of "spring" exercises every day.

At both hospitals one simple piece of apparatus made all the difference: a handle over the head of the bed meant that I could at least partially turn myself in bed and it also gave me a splendid thing to grip when severe pain swept over me.

Long recovery

Nights continued to be especially bad, though I was free to ask for analgesics if pain was severe. It was about 10 more weeks before I could go through a night without an injection of pentazocine and I needed more than one injection on many nights. What a difference that made to comfort and relief when compared with the rigid regimes in some hospitals: "Not within 4 hours," or "No hypnotic after 2 am." Hours are just as long between 3 am and 7 am as at any other time and the pains of peripheral neuritis are most severe in the night and early morning.

Twelve weeks after the onset of the syndrome I could just stand with support and use my arms to swing myself into a chair. What a thrill it was when after 15-16 weeks I could control my hands enough to be able to wipe my own bottom. By about 20 weeks I had acquired enough strength and co-ordination to be able to put letters into a crossword puzzle.

I recall being encouraged to get down on to the floor and then to crawl taking my first faltering steps—knowing where my legs were without looking—first starting to walk up and down stairs, being able to shave and wash myself, and hosts of other achievements great and small. I was supported by the patient dedication of the nurses, who, daily and sometimes several times a day, took me for walks up and down stairs and along corridors.

Throughout my illness I received the utmost help, understanding, and encouragement from everyone—starting with the neurologist, who told me to expect complete recovery. The nurses and physiotherapists never allowed me to feel a burden. I was encouraged to do as much for myself as I could, to attempt jigsaw puzzles and play cards, but I found that ataxic hands made these activities very frustrating.

I had a constant stream of visitors, though this was very tiring in the early stages of my illness. Medical and nursing colleagues talked enough shop to maintain professional interest and stimulation; sometimes they asked my opinion. But the weakness and ataxia were at times almost unbearably frustrating and the sensory loss, particularly that of positional sense, very bizarre.

Going home

My first outing was in March 1975, when my wife took me for a drive. It was marvellous to see ordinary events—people at work, cars, children at play. The most vivid sensation was one of speed: we seemed to be going so fast at 30 mph, and I remembered then how fast the ambulance had seemed to travel between hospitals. I spent a night at home towards the end of March and another two nights over Easter. By this time I could get myself up and down stairs with a crutch and into and out of the bath unaided. Between leaving hospital on 26 April and returning to work on 30 June I attended the physiotherapy department five days a week. I discarded my elbow crutches comparatively quickly and got along slowly with two sticks. I walked progressively further, started to do simple jobs in the garden while sitting on a box or stool, and did what I could indoors—washing up, peeling potatoes, etc. But I still needed prolonged periods of rest and was troubled by oedema of the feet and ankles.

I returned to limited work on 30 June, my working day being arranged round the daily physiotherapy session at 9·30. Ward visiting had to be limited by the sheer distance but I could see patients by appointment and conduct a reduced weekly outpatient clinic. Note writing was tedious and slow and dictating machines difficult to use. Pressing switches and knobs requires a surprising amount of finger power. The lack of independence was frustrating, and even now I have not rejoined the night or weekend rota.

Walking became steadily, if gradually, easier and less ungainly. By November 1975 I could walk without a stick in good conditions. I still tend to look down when walking and to "slap" my feet but I could walk on a narrower base than before and did not lurch so much. My balance continued to improve with increasing return of power and proprioceptive sensation in my ankles. By this time proximal power was about normal in my arms and legs but dorsiflexion of my ankles was still limited. My left hand was more or less normal in shape, although the lumbricals and interossei were still wasted and my little finger remained a little "hooked." My right hand was very weak and extremely wasted and I had very limited power in the fingers. I could extend the ring and little fingers with difficulty. My hand was usually steady enough to write, and if I was comfortable and using a firm table or desk my writing was fairly good. As long as the ulnar border of my hand was wasted, I had to fit my hand to a small piece of paper which would slide across the surface when I wanted to write, because I did not have the power or the necessary sensory innervation to move the hand myself. Signing letters remains laborious and time-consuming.

Setback

On 27 November 1975 I went for an early morning walk using only one stick. I lurched, caught my foot in a grating, and fell. I thought I had bruised myself, but when I got indoors I found that I could not get upstairs. A radiograph showed an intertrochanteric fracture of the left femur, and I was again admitted to hospital, where the orthopaedic surgeon inserted a pin and plate, and after three days I returned to the local hospital. I continued physiotherapy and had to walk entirely on crutches for three weeks. On 20 December, after I had begun walking again, I returned home and I started work on 5 January 1976.

Postscript

Now, over two years after the onset, my balance and walking are much improved and walking with a stick is "almost normal." My hands remain weak and wasted but steadier.

Sensation remains severely distorted and impaired. Positional sense is almost normal and I can even find notes on the piano without looking. Paraesthesiae of both hands is continuous and makes fine touch difficult, although gross touch is now better. The soles of my feet remain extremely sensitive and stepping even on the edge of the bath mat feels like treading on a steel wire. Perhaps the most unpleasant disturbance, however, is temperature sensation. For a time I lost all sense of temperature and would complain of feeling cold when others felt warm. This happens rarely now but I do have odd and changeable feelings, both subjective and objective, of cold in my fingers and feet. Subjectively the coldness is so intense as to be painful. Difficulty in starting or finishing micturition is still sometimes a problem. I can drive without difficulty.

I still waken in the early morning with pain in my legs, especially the knees and calves, and these pains will often come on at other times, usually towards evening. Sitting for long in any one chair is uncomfortable and with wasted muscles I have to have adequate cushions on most chairs. Another extremely unpleasant pain which occurs intermittently is a feeling that the os calcis or the big toe is being squeezed in a vice.

Residual symptoms occur in some patients who have suffered the Guillain-Barré syndrome—usually in those who showed little or no progress in the first two to three weeks. I often wonder whether my colleagues are being unduly optimistic about my ultimate full recovery, but time alone will tell.

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Today's Treatment

Diseases of the urinary system

Urinary tract infections

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Over the past 40 years numerous antibacterial agents effective in the treatment of urinary tract infections (UTI) have been introduced yet morbidity from these common non-obstructive infections continues to be a major problem. Up to 59 per 1000 consultations in general practice are for symptoms suggesting UTI. The founding of lay organisations that seek a better deal for sufferers illustrates our therapeutic shortcomings. Factors that contribute to the continuing morbidity include our inability to eradicate the source of urinary pathogens; the existence of a large reservoir of covert infections in the healthy population; the frequency of recurrent infection after treatment, and, the most taxing problem of all, the occurence of symptoms similar to those of UTI in the absence of infection —a problem that falls outside the scope of this article.

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Prophylaxis

The commonest source of urinary pathogens is the bowel flora, and the organisms that cause UTI do not possess special pathogenicity. Therefore eradication of the source of UTI requires elimination of the normal bowel flora. This is not feasible on a long-term basis. Prophylaxis by preventing the entry of micro-organisms into the urinary tract holds out greater promise. The value of aseptic precautions during instrumentation of the urinary tract and closed catheter drainage is now common knowledge. Since colonisation of the introitus in the female and subprepucial space in the male precedes UTI, improvement of perineal hygiene or local use of antibacterial agents, or both, might be expected to prevent infection. So far there have been no controlled studies to establish the value of such measures.

Quantitative urine culture enabled the identification of covert infections in apparently healthy populations. These infections are common (table 1), and they predispose to symptomatic infection. Screening for covert infections might therefore be expected to reduce morbidity from UTI. This is certainly so in pregnancy. Some 30% of pregnant women with covert bacteriuria develop acute pyelonephritis and eradication of the bacteriuria in early pregnancy can prevent this. In non-